# Significance of blood aspiration in carotid artery stenting with Angioguard XP

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***Background and Purpose:*** **In some patients, angiographic flow impairment is observed during carotid artery stenting (CAS) using Angioguard XP (AGXP), resulting in neurological symptoms. CAS was thus modified to improve clinical outcome.**

***Methods:*** **Ninety-seven patients were treated with CAS using AGXP from January 2008 to October 2009. In period I (January-December 2008; n = 53), blood aspirations were performed only in no-flow cases. In period II (January-October 2009; n = 44), blood aspirations were performed in no-flow and slow-flow cases. Clinical outcome, detection of microembolic lesions on diffusion-weighted imaging (DWI) and flow impairment during CAS were examined between these two periods before and after modifying the CAS procedure.**

***Results:*** **Periprocedural transient ischemic attacks occurred in 10 patients (18.9%) and one patient (2.27%) in periods I and II, respectively ($P$ = .018). Minor and major strokes were observed in two patients in each period ($P$ = .849). New ipsilateral DWI lesions were detected in 25 patients (47.2%) and 11 patients (25.0%) in periods I and II, respectively ($P$ = .024). Among 18 slow-flow cases, new DWI lesions were detected in one patient (9.09%) and five patients (71.4%) with (n = 11) and without (n = 7) blood aspirations, respectively ($P$ = .013). Neurological symptoms were observed only in three of seven patients (42.9%) without aspirations, compared to one of 11 patients (9.1%) with aspirations ($P$ = .043).**

***Conclusion:*** **Postoperative symptomatic stroke and new DWI lesions are significantly associated with blood flow impairment during CAS using AGXP. When flow impairment occurs, blood aspiration should be performed. (J Vasc Surg 2011;53:1478-84.)**

Carotid artery stenting (CAS) has been established as an alternative procedure to carotid endarterectomy (CEA) since the Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy (SAPPHIRE) trials,[1,2] but embolic lesions detected on diffusion-weighted imaging (DWI) may occur more frequently in cases of CAS than in those of CEA. Whereas these lesions are almost silent, they can be associated with cognitive impairment found on neuropsychometric examination.[3-6]

Whereas CAS is performed using the Angioguard XP (AGXP; Cordis Endovascular, Miami Lakes, Fla), which is the only approved device for use during CAS in the Japanese Health Insurance System, angiographic flow impairment proximal to the filter may result in new cerebral embolic lesions and symptomatic stroke.[7,8] The clinical impact of blood aspiration on flow impairment is assessed by evaluating postprocedural symptomatic stroke and new embolic lesions on DWI, which are associated with flow impairment.

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## METHODS

**Patient population.** Ninety-seven patients in the Hyogo Brain and Heart Center at Himeji were treated with CAS using AGXP from January 2008 to October 2009. A total of 82 men and 15 women, with a mean age of 73.2 years (range, 57-85 years) were enrolled in the study. Based on the criteria used in the SAPPHIRE trials, CAS was indicated for patients having a high-risk factor for CEA and over 50% symptomatic stenosis or over 80% asymptomatic stenosis; stenosis was determined using the North American Symptomatic Carotid Endarterectomy Trial protocol criteria. CEA high-risk factors include previous CEA, previous neck surgery, high level C1 or C2 lesions, and severe active coronary artery disease (CAD). A carotid stenosis was considered symptomatic if the patient had experienced an ipsilateral ocular or cerebral ischemic event within 6 months before the start of the study. According to this criteria, 31 patients were labeled symptomatic and 66 asymptomatic.

The patients were grouped into two time periods: 53 patients (43 men, 10 women) with a mean age of 72.8 years (range, 57-84 years) were treated from January to December 2008 (period I), and 44 patients (39 men, five women) with a mean age of 73.8 years (range, 60-84 years) were treated from January to October 2009 (period II). The clinical characteristics of the patients are presented in Table I.

Cerebral angiograms were evaluated for a definitive diagnosis of the lesions. Preoperative and postoperative degrees of carotid stenosis, lesion length, and the number of stents were determined using the angiographic data are presented in Table I.

**Table I.** Clinical and lesion characteristics in both groups of patients

| | *Period I* (*n = 53*) | *Period II* (*n = 44*) | P value |
|---|---|---|---|
| Clinical characteristics | | | |
| Men | 43 (81.1%) | 39 (88.6%) | .31 |
| Age (years) | 72.8 ± 6.3 | 73.8 ± 7.0 | .42 |
| HTN | 47 (88.7%) | 32 (72.7%) | .045[a] |
| DM | 25 (47.2%) | 20 (45.5%) | .87 |
| Hyperlipidemia | 20 (37.7%) | 19 (43.2%) | .74 |
| CAD | 21 (39.6%) | 19 (43.2%) | .73 |
| Symptomatic | 14 (26.4%) | 17 (38.7%) | .2 |
| History of TIA | 4 (7.5%) | 9 (20.5%) | .06 |
| History of cerebral infarction | 19 (35.8%) | 13 (29.5%) | .52 |
| Lesion characteristics | | | |
| Degree of stenosis (%) | | | |
| Pre-CAS | 80 ± 9 | 81 ± 11 | .550 |
| Post-CAS | 6 ± 8 | 9 ± 8 | .07 |
| Lesion length (mm %) | 19.7 ± 11.2 | 19.9 ± 12.9 | .96 |
| Ulceration | 23 (43.4%) | 11 (25.0%) | .060 |
| Procedural characteristics | | | |
| Number of stents used | 1.16 ± 0.37 | 1.27 ± 0.60 | .27 |
| Fluoroscopy time (minutes) | 25.7 ± 3.00 | 25.1 ± 2.70 | .29 |

*CAD,* Coronary artery disease; *CAS,* carotid artery stenting; *DM,* diabetes mellitus; *HTN,* hypertension; *TIA,* transient ischemic attack.
Continuous variables are shown as means with SDs in parentheses.
Categorical variables are shown as frequencies with percentages in parentheses.
[a]$P < .05$.

Two days before CAS, magnetic resonance imaging (MRI) was routinely performed using a 3T MR Imager (Intra; Philips Medical Systems, Best, The Netherlands) followed by sequencing of T1-weighted and T2-weighted images, fluid-attenuated inversion recovery imaging, DWI, and time-of-flight MR angiography.

Written informed consent for diagnostic and interventional procedures was obtained from each patient.

**Data collection.** Clinical, radiological, and procedural data were obtained for each patient. Clinical variables included the following: age, gender, hypertension (blood pressure ≥140/90 mm Hg measured repeatedly or presence of antihypertensive drugs), diabetes mellitus (HbA1c >6.5%, fasting blood glucose >120 mmol/L or presence of antidiabetic drugs), hyperlipidemia (fasting serum cholesterol levels >220 mmol/L), and CAD (history of angina, myocardial infarction, percutaneous transluminal angioplasty, or surgery). Radiological variables were established by the evaluation of postprocedural MRI and digital subtraction angiography (DSA). These variables included the number of postprocedural embolic lesions in DWI, grade of stenosis, and the presence of ulcerated plaques on both DSA and ultrasonography. Lesion ulceration was defined as a recess having a depth of 2 mm or more from the plaque surface.

**Carotid artery stenting.** Administration of two types of oral antiplatelet agents, including aspirin (100 mg, n = 97) in combination with clopidogrel (75 mg, n = 95), cilostazol (200 mg, n = 1), or ticlopidine (200 mg, n = 1) was initiated at least 4 days before CAS. General anesthesia was induced for 13 patients with occlusion of the contralateral carotid artery and poor contralateral vessels.

After intravenous administration of 100 IU/kg heparin to achieve an activated clotting time (ACT) of over 300 seconds, an 8F guiding catheter was inserted through an 8F-long sheath in the femoral artery and was placed in the common carotid artery. The lesions were bypassed using AGXP and the filter was opened. Predilatation with 3.0-mm-diameter balloon was indicated for the lesions with high stenosis rate or calcification in 56 of 97 patients (57.7%). This was followed by implantation of the PRECISE stent (Cordis Endovascular), with postdilatation of lesions for complete recovery. Carotid flow was angiographically after each step with manual injections (4 mL, 2 mL/s).

Flow impairment was determined on an angiogram taken immediately after postdilatation. The thrombolysis in myocardial infarction (TIMI) flow grade classification for the coronary artery was modified[9] to define the flow impairment in the carotid artery:

- No flow was defined as flow arrest corresponding to grade 0 in TIMI, or that the contrast agent could go through the filter but could not reach the middle cerebral artery.
- Slow flow was defined as flow reduction with contribution to the middle cerebral artery, corresponding to grade 2 in TIMI.
- Normal flow corresponded to grade 3 in TIMI.

Where flow impairment was observed, multiple aspirations of the blood column proximal to the filter were performed using the Export aspiration catheter (Medtronic, Minneapolis, Minn) and a 30-mL syringe, around the distal edge of the stent proximal to the filter, which was at least 20 mm away from the filter basket. Aspirated blood was filtered through the four-folded two-ply gauze. There was usually some debris left on the gauze. Blood aspirations were performed until debris could not be macroscopically observed. Neurological symptoms and vital signs were monitored during the multiple aspirations in each patient by the operator and the assistants. The filter was finally retrieved using a capture sheath. Multiple aspirations were indicated for no-flow cases during period I and for both no-flow and slow-flow cases during period II.

An MRI was routinely performed about 48 hours after CAS to detect new ischemic lesions, not seen on the pre-CAS MRI.

**Definitions of complications after carotid artery stenting.** The primary clinical outcome of this study was evaluated with respect to incidence death, myocardial infarction, or neurological symptoms, including transient

**Table II.** Outcomes of neurological symptoms and DWI lesions after CAS in slow-flow, no-flow, and normal-flow cases in each period

| | *Neurological symptoms* | | *DWI lesions* | |
|---|---|---|---|---|
| | *TIA* | *Minor/major stroke* | *Positive on DWI* | *Mean number of microemboli* |
| Period I | | | | |
| Normal flow (n = 36) | 3 (8.33%) | 1 (2.78%) | 15 (41.7%) | 1.31 ± 2.52 |
| Slow flow (n = 7) | 3 (42.9%) | 0 | 5 (71.4%) | 2.71 ± 2.56[b] |
| No flow (n = 10) | 4 (40.0%) | 1 (10.0%) | 5 (50.0%) | 3.00 ± 5.50 |
| Subtotal (n = 53) | 10 (18.9%)[a] | 2 (3.77%) | 25 (47.2%) | 1.75 ± 3.21[c] |
| Period II | | | | |
| Normal flow (n = 29) | 0 | 2 (6.90%) | 9 (31.0%) | 1.28 ± 2.34 |
| Slow flow (n = 11) | 0 | 0 | 1 (9.09%) | 0.18 ± 0.60[b] |
| No flow (n = 4) | 1 (25.0%) | 0 | 1 (25.0%) | 0 |
| Subtotal n = 44 | 1 (2.27%)[a] | 2 (4.55%) | 11 (25.0%) | 0.89 ± 1.99[c] |

*CAS*, Carotid artery stenting; *DWI*, diffusion-weighted imaging; *TIA*, transient ischemic attack.
Continuous variables are shown as means with SDs in parentheses.
Categorical variables are shown as frequencies with percentages in parentheses.
TIA: any new neurological deficit that was resolved within 24 hours.
Minor stroke: any new neurological deficit that persisted for more than 24 hours and was resolved completely within 30 days.
Major stroke: any new neurological deficit that persisted after 30 days and increased the modified ranking scale ≥1 point.
[a]$P = .010$.
[b]$P = .006$.
[c]$P = .024$.

ischemic attack (TIA) and minor and major stroke within 30 days after CAS. Each patient was neurologically examined prior to CAS, the day after CAS, and at day 30. The definitions of neurological complications that occurred within the first 24 hours and within the next 30 days after CAS were defined as follows based on a previous study by Mathur[10]:

- TIA: any new neurological deficit that was resolved within 24 hours.
- Minor stroke: any new neurological deficit that persisted for more than 24 hours and was resolved completely within 30 days.
- Major stroke: any new neurological deficit that persisted after 30 days and increased the modified Ranking Scale ≥1 point.

Neurological deficit, which emerged predilatation or postdilatation and disappeared after deflation of the balloon, was not defined as TIA.

**Statistical analysis.** Continuous and categorical variables were reported as means with SDs and frequencies with percentages, respectively. The clinical characteristics and procedural and radiological variables were compared between patients in periods I and II and between patients with flow impairment and those with normal flow. The comparisons were made using the independent samples t test, $\chi^2$ test with Yates correction, or the Fisher exact test. Among slow-flow cases, patients with multiple aspirations were compared to those with no aspiration using the $\chi^2$ test with Yates correction or the Fisher exact test. A *P* value less than .05 was considered statistically significant.

## RESULTS

Apart from hypertension, no significant differences were observed in patients and lesional characteristics (Table I) between the two time periods. No patient died or suffered myocardial infarction for 30 days after CAS.

Neurological symptoms were observed in 15 patients: 12 patients during period I, which included 10 TIAs, one minor stroke, and one major stroke, and three patients during period II, which included one TIA and two minor strokes ($P = .35$; Table II). Incidence of TIA was significantly reduced in period II ($P = .010$; Table II). In period I, positive DWI lesions were seen in five of 10 no-flow cases (50%), five of seven slow-flow cases (71.4%), and 15 of 36 normal-flow cases (41.7%; Table II). In period II, positive DWI lesions were seen in one of four no-flow cases (25%), one of 11 slow-flow cases (9.1%), and nine of 29 normal-flow cases (31.0%; Table II). Periprocedural DWI lesions in slow-flow cases were significantly reduced in period II ($P = .006$; Table II).

TIA was resolved within 30 minutes after each CAS procedure. All three patients with minor strokes experienced upper extremity numbness for 1 week after the procedure. One patient with major stroke experienced left hemiparesis, which worsened from grade 0 to grade 2 on the modified Rankin scale after 1 month. Neurological symptoms were observed in 15 patients; after postdilatation in 10 patients, after placement of the stent in one patient, and after predilatation in one patient. In the remaining three patients, two complained of numbness in their upper limbs and one complained of a mild disturbance in finger movement after CAS. The exact time of onset was uncertain.

Total fluoroscopy times for the procedure were 25.9 ± 2.2 minutes and 25.2 ± 3.1 minutes in flow impairment and normal flow, respectively ($P = .77$; Table III). The mean time between the CAS procedure and the follow-up MRI was 51 hours.

**Table III.** Clinical and lesion characteristics of slow-flow or no-flow and normal-flow cases

| | *Slow or no flow* (*n* = 32) | *Normal flow* (*n* = 65) | P value |
|---|---|---|---|
| Clinical characteristics | | | |
| Men | 31 (96.9%) | 51 (78.5%) | .018[a] |
| Age (years) | 73.3 ± 6.1 | 73.3 ± 6.9 | .998 |
| HTN | 26 (81.3%) | 53 (81.5%) | .973 |
| DM | 14 (43.8%) | 31 (47.7%) | .718 |
| Hyperlipidemia | 12 (37.5%) | 27 (41.5%) | .872 |
| CAD | 10 (31.3%) | 30 (46.2%) | .164 |
| Symptomatic | 13 (40.6%) | 18 (27.7%) | .203 |
| History of TIA | 4 (12.5%) | 9 (13.8%) | .857 |
| History of cerebral infarction | 13 (40.6%) | 19 (29.2%) | .266 |
| Lesion characteristics | | | |
| Degree of stenosis (%) | | | |
| Pre-CAS | 83 ± 10 | 79 ± 10 | .090 |
| Post-CAS | 7 ± 7 | 8 ± 8 | .813 |
| Lesion length (mm) | 22.7 ± 16.4 | 18.4 ± 8.8 | .094 |
| Ulceration | 16 (50.0%) | 18 (27.7%) | .031[a] |
| Procedural characteristics | | | |
| Number of stents used | 1.28 ± 0.68 | 1.18 ± 0.39 | .378 |
| Fluoroscopy time (minutes) | 25.9 ± 2.2 | 25.2 ± 3.1 | .772 |

*CAD,* Coronary artery disease; *CAS,* carotid artery stenting; *DM,* diabetes mellitus; *HTN,* hypertension; *TIA,* transient ischemic attack.
Continuous variables are shown as means with SDs in parentheses.
Categorical variables are shown as frequencies with percentages in parentheses.
[a]$P < .05$.

**Table IV.** Radiological and neurological symptoms after CAS in slow-flow or no-flow and normal-flow cases

| | *Slow or no flow* (*n* = 32) | *Normal flow* (*n* = 65) | P value |
|---|---|---|---|
| Outcomes | | | |
| Presence of ipsilateral microemboli | 12 (37.5%) | 24 (36.9%) | .957 |
| Mean number of microemboli | 1.50 ± 3.34 | 1.29 ± 2.43 | .728 |
| Neurological symptoms | 9 (28.1%) | 6 (9.23%) | .019[a] |
| TIA | 8 (25.0%) | 3 (4.62%) | .005[a] |
| Stroke | 1 (3.13%) | 3 (4.62%) | .845 |
| Minor stroke | 0 | 3 (4.62%) | .221 |
| Major stroke | 1 (3.13%) | 0 | .155 |

*CAS,* Carotid artery stenting; *TIA,* transient ischemic attack.
Continuous variables are shown as means with SDs in parentheses.
Categorical variables are shown as frequencies with percentages in parentheses.
TIA: any new neurological deficit that was resolved within 24 hours.
Minor stroke: any new neurological deficit that persisted for more than 24 hours and was resolved completely within 30 days.
Major stroke: any new neurological deficit that persisted after 30 days and increased the modified ranking scale ≥1 point.
[a]$P < .05$.

Eleven of 15 patients (73.3%) with neurological symptoms had postoperative DWI lesions. New postoperative lesions on DWI were detected in 25 patients (47.2%) during period I and 11 patients (25.0%) during period II (Table II), a number significantly lower than that in period I ($P = .024$). The number of lesions were 1.75 ± 3.21 and 0.89 ± 1.99 in periods I and II, respectively ($P = .131$).

Overall, 12 of 36 patients (33.3%) with postoperative DWI lesions had neurological symptoms. The number of lesions were 5.08 ± 4.77 in patients with neurological symptoms and 2.65 ± 2.08 in those without symptoms, respectively ($P = .04$) and 1.45 ± 1.75 in the patients with TIA, and 10.8 ± 4.65 in patients with minor or major stroke, respectively ($P < .0001$). Increased lesion load was significantly related to neurological symptoms. Out of 36 patients with DWI lesions, 33 patients (92%) showed ipsilateral lesions, whereas three patients (8%) had bilateral lesions. There were no lesions observed in the vertebral artery region. Occlusion of the main cerebral branches could not be observed on post-CAS angiograms in all patients.

On the other hand, three of 61 patients (4.92%) without postoperative DWI lesions had neurological symptoms ($P < .05$). In these cases, neurological symptoms emerged with the no-flow phenomenon and disappeared after the CAS procedure. The symptoms were probably associated with intolerance to the reduction of cerebral blood flow.

All of 32 cases of flow impairment during CAS occurred postdilatation; 17 of 53 (32.1%) in period I and 15 of 44 (34.1%) in period II. Clinical and lesional characteristics of no-flow or slow-flow and normal-flow cases are shown in Table III. Male gender ($P = .018$) and lesion ulceration ($P = .031$) were significantly associated with asymptomatic flow impairment. As shown in Table IV, nine of 32 patients (28.1%) with flow impairment had neurological symptoms (eight TIAs and one major stroke), and six of 65 patients (9.23%) with normal flow had neurological symptoms (three TIAs and three minor strokes), indicating that flow impairment was significantly related to neurological symptoms ($P = .007$).

Out of 10 patients who underwent CAS within 1 month of the last reported symptoms, six patients (60%) showed DWI lesions. Out of 21 patients who underwent CAS after 1 month of the last reported symptoms, four patients (19.0%) showed DWI lesions ($P = .040$).

Slow flow was observed in seven and 11 patients during periods I and II, respectively (Table II). No neurological symptoms were observed in 11 patients with multiple aspirations (period II), but three of seven patients (42.9%) without aspirations had TIAs (Table V; $P = .043$). New DWI lesions were observed in one of 11 patients (9.09%) with multiple aspirations and five of seven patients (71.4%) without aspirations ($P = .013$). We could observe some macroscopic debris in the aspirated blood from all patients with slow flow during CAS (Fig 1). The mean number of blood aspirations was 7.7 (range, 4-17) in slow-flow cases.

**Table V.** Radiological and neurological symptoms after CAS in slow-flow cases with multiple or no aspiration

| | *Multiple aspirations* (*n* = 11) | *No aspiration* (*n* = 7) | P value |
|---|---|---|---|
| Outcomes | | | |
| Presence of ipsilateral microemboli | 1 (9.09%) | 5 (71.4%) | .013[a] |
| Mean number of microemboli | 0.18 ± 0.60 | 2.71 ± 2.56 | .006[a] |
| Neurological symptoms | 0 | 3 (42.9%) | .043[a] |
| TIA | 0 | 3 (42.9%) | .043[a] |
| Stroke | 0 | 0 | n.s. |
| Minor stroke | 0 | 0 | n.s. |
| Major stroke | 0 | 0 | n.s. |

*CAS,* Carotid artery stenting; *n.s.,* not significant; *TIA*, transient ischemic attack.

Continuous variables are shown as means with SDs in parentheses.

Categorical variables are shown as frequencies with percentages in parentheses.

TIA: any new neurological deficit that was resolved within 24 hours.

Minor stroke: any new neurological deficit that persisted for more than 24 hours and was resolved completely within 30 days.

Major stroke: any new neurological deficit that persisted after 30 days and increased the modified ranking scale ≥1 point.

[a]$P < .05$.

## DISCUSSION

CAS has been used as an alternative procedure to CEA worldwide since the SAPPHIRE trial proved the superiority of CAS over CEA. Major adverse events (MAEs) within 30 days, including minor and major strokes, death, or myocardial infarction, have an incidence of 4.8%[1,2] in CAS cases. In our study, MAEs were observed in four of 97 patients (4.12%). However, TIA, the most frequent neurological symptom observed in our study, which was not described in the SAPPHIRE trial, was the reason for the overall high incidence rate of neurological symptoms of 22.6% and 6.8% in period I and period II, respectively. TIAs were observed more frequently in flow impairment cases than in normal-flow cases in period I. A majority of periprocedural DWI lesions remain clinically silent, but neuropsychometric examination revealed cognitive dysfunction in some of the clinically silent cases.[6,11,12] Moreover, increased lesion load may be responsible for the clinical impact.[13,14]

Lesion ulceration and masculine gender were significant factors helping in the prediction of flow impairment. Lesion ulceration was related to vulnerable plaque, which is an independent risk factor for stroke,[15] and can be detected by advanced assessment on ultrasonography or MRI.[16,17] Lesion length, the percentage of stenosis, and the number of stents used were not related to the incidence of flow impairment. This suggests that the flow impairment may be associated with the vulnerability of the carotid plaque. The relation between masculine gender and flow impairment was uncertain, but 69 of 82 men (84%) were smokers and none of the women were smokers; this finding indicates that smoking is a risk factor for stroke and is related to vulnerable plaque, as Redgrave et al[18] demonstrated.

This study demonstrates a significant correlation between flow impairment and the occurrence of post-CAS neurological symptoms and DWI lesions. Furthermore, the number of DWI lesions significantly correlates with the severity of neurological symptoms. In period I, TIA occurred in 42.9% of slow-flow cases, which was higher compared to 40.0% in no-flow cases. In the same period, the frequency of DWI lesions was 71.4% in slow-flow cases, which was unexpectedly higher compared to 50.0% in no-flow cases. Based on these results, we modified the CAS procedures for period II, emphasizing on blood aspiration for slow-flow cases. As a result, blood aspiration reduced the frequency of neurological symptoms and DWI lesions in slow-flow cases.

Flow impairment during CAS was observed in 32 of 97 patients (33.0%) in this study, which also shows the rate of flow impairment ranging from 10.1% to 40.0% with various filter devices.[7,8,19] In this study, neurological symptoms and DWI lesions were observed more frequently in no-flow or slow-flow cases than in normal-flow cases. It has been previously reported by Casserly et al[7] that patients with slow flow are at higher risk of stroke than patients with normal flow (9.5% vs 1.7%).

The filter should ideally capture all the debris coming from the arterial wall and the various kinds of plaques, but it has several limitations. Instances where the volume of debris exceeds the filter's volume, a no-flow phenomenon occurs and the excess debris floats proximal to the filter in the carotid artery, which then requires removal by blood aspiration. In slow-flow cases, where the filter pores are incompletely occluded by the captured debris, the blood flow is disturbed but maintained. When the volume of the debris does not exceed that of the filter basket, the filter can capture all debris, which can be retrieved without aspiration. However, this explanation is only theoretic and cannot support the results of this study, including the high frequency of post-CAS neurological symptoms and DWI lesions in slow-flow cases during period I. Hence, a more practical approach is required.

The second limitation is the capture ability with respect to different kinds of debris. While rinsing the aspirated blood with heparinized saline on a gauze piece, two different kinds of debris were macroscopically observed: (1) small solid fragments corresponding to the vascular intima and parts of hard plaque and (2) yellowish-white fragile material, originating from the ruptured contents of the lipid-rich soft plaques (Fig 1). While solid fragments could be found within the filter basket (Fig 2), fragile materials were usually found on the gauze, because the size of the solid fragments was larger than the size of the fragile material. One of the explanations about the material is that it could not be debris but thrombus formed in stagnated blood flow proximal to the filter. But to maintain the ACT, ≥300 seconds with heparinization and almost the same fluoroscopy time between no-flow or slow-flow and normal flow, do not support the explanation.

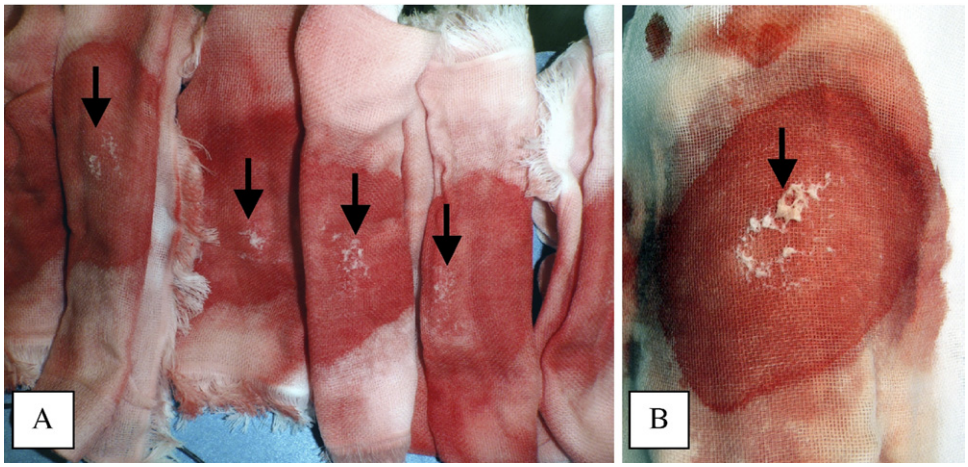


**Fig 1.** Debris left on the gauze from aspirated blood in a slow-flow case. Blood aspiration was performed six times in this case. **A,** Debris (*arrow*) was found four times in six aspirations. **B,** The appearance of debris left on the gauze (*arrow*) was viscous and fragile.

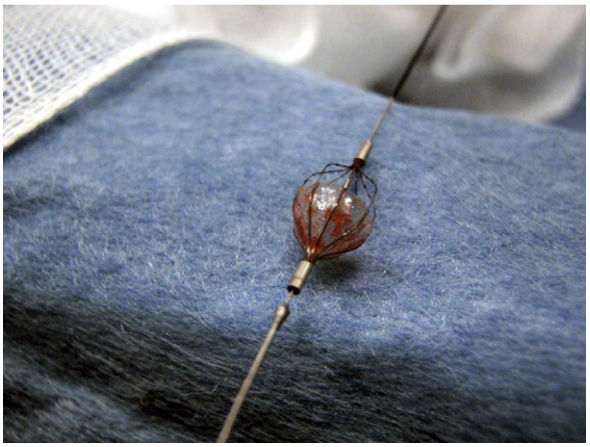

**Fig 2.** Captured debris in the filter of the Angioguard XP. The appearance of debris was solid.

Our results indicate that the AGXP may be incapable of capturing the fragile debris floating within the internal carotid artery in slow-flow cases. As a result, failure to perform blood aspiration in the period I cases led to a higher frequency of post-CAS neurological symptoms and DWI lesions, the frequency of which decreased with blood aspiration in period II. According to Maulex et al,[5] the presence of debris captured by the filter device could not be significantly related to new DWI lesions after CAS, indicating the presence of some different kinds of debris in the internal carotid artery. Casserly et al[7] examined the aspirated blood in flow impairment cases and found that the debris consisted of lipid-laden macrophages, cholesterol clefts, and fibrin materials, which could correspond to the fragile materials in our study, indicating that multiple blood aspirations should be performed in flow impairment cases. A detailed examination of other prospective filter devices should be carried out to assess their capture volume and capture ability for various kinds of debris.

## CONCLUSION

Flow impairment could be caused by the debris originating from the core of carotid plaques. Some kinds of debris could not be captured completely with the AGXP filter, which resulted in neurological symptoms and DWI lesions. Multiple blood aspirations proximal to the filter in case of flow impairment significantly reduced perioperative TIAs and new DWI lesions after CAS with the AGXP device, hence this procedure is recommended to reduce TIAs and DWI lesions in flow-impairment cases.

## AUTHOR CONTRIBUTIONS

Conception and design: TM, MN
Analysis and interpretation: TM, MN
Data collection: TM, MN, YM, YU, MS, SO
Writing the article: TM
Critical revision of the article: TM, MN
Final approval of the article: TM, MN
Statistical analysis: TM
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Overall responsibility: TM

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